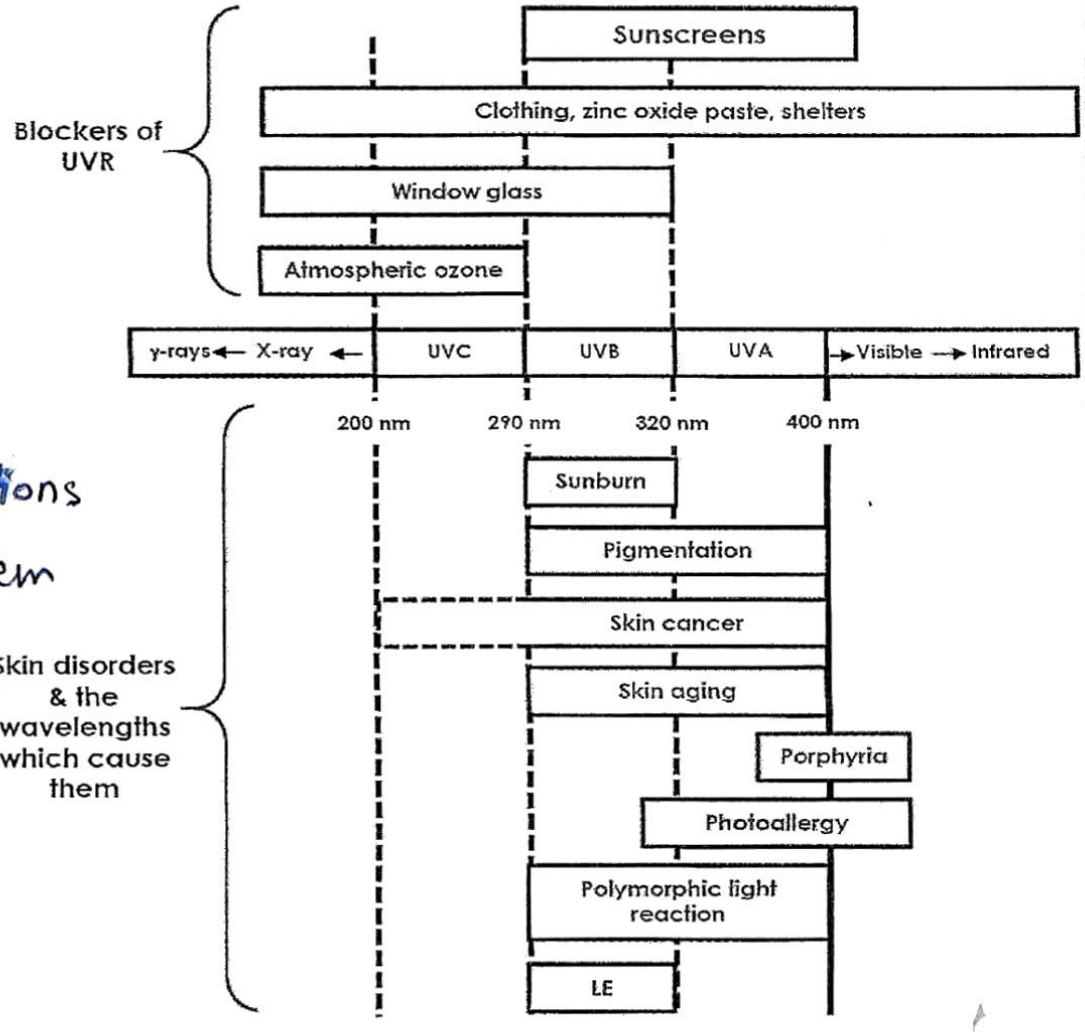
Dermatoses Due to physical agents

- Ultraviolet light
- photodermatoses
  - ↳ polymorphus light eruption
  - ↳ Actinic prurigo
  - ↳ Chronic actinic Dermatitis
  - ↳ Actinic Reticuloid
  - ↳ Solar Urticaria
  - ↳ solar elastosis
  - ↳ Nodular elastosis & Cysts and Comedones
- photosensitivity
- phytophotodermatitis
- photoprotection
  - ↳ Sunscreens
- Diseases aggrevated By Cold.
  - ↳ Frost Bite
  - ↳ pernio Chilblains
  - ↳ Radiodermatitis
  - ↳ Erythema abigne
  - ↳ Immersion Trench Foot

Amylee

1 Ultraviolet Light

- Exposure of the skin to UV light has:
  - Acute short term effect
  - Chronic long term effect
  (Both - wavelength Dependant)
  - profound effect è non-erythromagenic Doses of UV light
- UV light induce several Diffrent Types of DNA Damage as pyrimidine Dimers
  - oxidative guanine Base modifications
- UV light affect :- the skin's immune system exerting Both → pro
  → anti-inflammatory Response
- Several DNA Repair pathways are involved in the processing of UV-light-induced DNA Damage.
  - → nucleotide excision Repair
  - → Base excision Repair
  - → translesional DNA synthesis
  - → Recombination Repair



- The solar spectrum at sea level includes :-
  - wavelength of about 290 - 3000 nm
- The **Ozone** in the upper atmosphere provides :-
  Barrier to wave lengths shorter than 290nm
  ↓
  So the earth's surface → Shielded from the shorter wavelengths
  ↓
  which are → inimical to most forms of life
- The sunlight which Reaches ground level :-
  Consist of :-
  - UV Radiation from 290 to 400 nm
  - visible radiation from 400 to 700 nm
- Sun's UV Radiation:
  → UVA > 95%.
  → All of the UVC and much of UVB → absorbed By the oxygen and ozone.

UVR	UVC	= 200-290 nm – germicidal reaction. They do not reach the earth's surface.
	UVB	= 290-320 nm (erythematogenic radiation). Some authors use 315 nm, rather than 320 nm, as the demarcation between UVA and UVB.
	UVA	= 320-400 nm (**UVA1** "340-400 nm" and **UVA2** "320- 340 nm").
Visible light		= 400-760 nm.
Near infrared Far infrared Microwaves & radiowaves		760-1,000 nm 1,000-100,000 nm $>10^6$

- Normal Response of human Skin to Solar irradiation :-

Acute Reactions

1 Erythema :- (sunburn)

- Caused By → UVB
- after sun exposure → onset of Erythema occur within → 2 to 6 hrs
- max intensity occur → 15 to 24 hr after exposure
- Minimum Erythema Dose (MED) = is the minimal amount of energy required to produce a uniform, clearly defined erythemal Response usually at 24 hrs.
- The ability of UV to induce sunburn Rapidly declines ∝ increasing of wavelength

→ **UVA** can cause Erythema But The Dose Required is much greater.

2 Pigmentation :-

Immediate
- By UVA and visible light
- U. Soon after exposure
- Lasts only few hrs
- D.t photo-oxidation of preuiously formed melanin

Delayed
- By UVB
- D.t formation of new melanin
- occur few days after exposure
- It protect skin from subsequent exposure

3 ↑↑ thickness of Epidermis

- occur few days after UVB or UVC exposure.
- It protect against subsequent exposure

4 Immunologic alteration

- Cellular and molecular events in UV irradiation skin may be:-

- mediating Pro-inflammatory immune Stimulating effect

- release of proinflammatory mediators By resident, non-resident skin Cells e.g:
  - Serotonin
  - Prostaglandin
  - IL-1, IL-6, IL-8
  - TNF α
- Induction of antimicrobial peptides

(Hypothesized to explain why UV-irradiation skin is Not prone to Bacterial infection)

- mediating Anti-inflammatory Immunosuppressive effect

- Depletion of langerhans cells or modulation of their antigen-presenting function
- Release of anti-inflammatory mediators By resident and non-resident skin cells e.g:
  - IL-10
  - α-MSH
- Induction of regulatory T-Cells (antigen-specific)

5 Excision repair:

UVR → Cause Damage of DNA → Followed By Excision repair which is a mechanism mediated By:- endonuclease enzyme

Action spectra for photodamage

Effect	Spectrum of light		
	UVB	UVA	Visible
Sunburn	++++	+	
Photoaging	++++	++	?
Squamous cell carcinoma	++++	+	
Basal cell carcinoma	+++	?	
Cutaneous melanoma	++	+	
Photoimmune suppression	++++	++	
Photosensitivity	+	+++	+

⊛ Chronic Reactions ⊛ [chronic actinic Damage]

- occur in: Blue eyes - Fair complexioned persons (sailor's sign - Farmer's Skin)

photoaging (premature aging)

- changes in apperance and function of the skin
- as a Result of → repeated exposure rather than passage of time alone
- e.g: Coarseness, mottled pigmentation, solar elastosis Telangectasia, purpura. fibrotic depigmented areas (pseudoscars)
- The only other enviromental factor that produce premature aging of skin is: **Cigarette Smoking**
- Treatment: 0.1% tretinoin Cream

Photo Carcinogenesis

(premalignancy - malignancy)

- e.g: Solar Keratosis
  - Bowen's Disease
  - Basal cell epithelioma
  - Sq. Cell carcinoma
  - melanoma
  - actinic chelitis
- disseminated superficial actinic porokeratosis

Photocarcinogenesis Cascade

UV → induced DNA Damage generates → mutations → accumulation of a sufficient number of mutation in Critical genes → malignant Transformation of single Keratinocyte or melanocyte → Formation of skin Cancer

Cutaneous signs of significant photodamage

1. Solar elastosis
2. Nodular elastosis
3. weathering nodules
4. Cutis rhomboidalis nuchae
5. Colloid milium
6. Cutan. melanoma (lentigo maligna)
7. Solar lentignes: ephelides
8. Squamous Cell Carcinoma
9. Solar purpura - hemosiderin deposition
10. Aquired elastotic hemangioma
11. Actinic keratosis
12. Actinic Comedonal plaque
13. Basal cell carcinoma
14. Pseudoscars
15. Poikiloderma of Civatte
16. Grover's Disease

[2] Photodermatosis :. Skin Disorders Induced OR excagerated By light .

Classification of photodermatoses

Immunologically mediated photodermatoses	Drug- & chemical-induced photosensitivity
• Polymorphous light eruption. • Chronic actinic dermatitis. • Actinic prurigo. • Hydroa vacciniforme. • Solar urticaria.	***Exogenous:*** • Phototoxicity: Systemic & topical. • Photoallergy: Systemic & topical. ***Endogenous:*** • Cutaneous porphyria.
Hereditary photodermatoses	**Photoaggravated dermatoses**
Caused by defects in nucleotide excision repair: • Xeroderma pigmentosum. • Cockayne syndrome, including cerebro-oculo-facio-skeletal syndrome. • Trichothiodystrophy. • Ultraviolet light sensitive syndrome. ***Caused by double strand break repair defects:*** • Rothmund-Thomson syndrome. • Bloom syndrome. ***Caused by abnormal chemical substances:*** • Smith-Lemli-Opitz syndrome. • Hartnup disease. ***Others:*** • Kindler syndrome.	• Atopic dermatitis. • Darier-White disease. • Dermatitis herpetiformis. • Herpes simplex infection. • Lupus erythematosus & neonatal lupus erythematosus. • Juvenile dermatomyositis. • Pellagra. • Psoriasis.

- **Laboratory Evaluation of photosensitivity :-**

1- Routine histology
2- Anti-nuclear antibodies
3- Anti-RO/SSA La/SSB antibodies
4- plasma porphyrins → followed By Complete porphyrin profile → if +ve
5- photoTesting to UVA, UVB, visible light
6- photopatch testing
7- in infants and children → Urinary amino acids

5

- **DD of photodermatoses most Commonly associated with different age groups :-**

1- when the ptn is a **Child** :-
- Jurenile spring eruption
- Childhood porphyrias
  - ↳ erythropoietic protophyria
  - ↳ Congenital erythropiotic protophyria
- Actinic prurigo
- Hdroa vaciniforme
- Genodermatoses

2- when the ptn is an **adult** :-
- polymorphous light eruption
- Drug-induced photosensitivity
- Solar Urticaria
- lupus erythematosus
- porphyria Cutanea Tarda

3- when the ptn is **old** :-
- Chronic actinic Dermatitis
- Drug induced photosensitivity
- Dermatomyositis

④ Phototesting :

- Confirm the presence of a photosensitivity Disorder
- most helpful for the Diagnosis of Immunologically-mediated photodermatosis
- using : opaque template with several windows
  - the uninvolved skin of the Back or abdomen → exposed to varying doses of UVA-UVB or visible monochromic or Broad-spectrum radiation.
- Following light exposure → the first reading : performed in 20 minutes to → Detect urticarial lesions as seen in Solar Urticaria

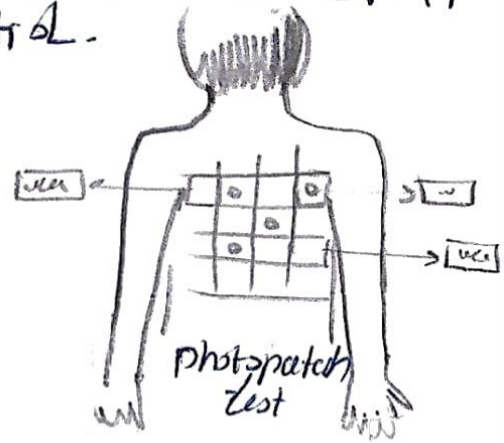
⑤ Photopatch Testing :

- evaluate pts è photoallergic Contact Dermatitis
- it's similar to standard patch test used for evaluation of allergic Contact Dermatitis
- Notable Difference include:

irradiation of the patch site è UVA in the Photopatch testing

- Duplicate sets of photoallergen panels are placed on the Back , and the sites are then Covered è an opaque material → to protect them from exposure to light.

- After 24hr → one of the panels is irradiated è a Dose of $10\ J/m^2$ of UVA OR 50 % of MED-A The other pannel act as Control.



Expected phototest & photopatch test results*

Disorder	MED for UVA	MED for UVB	Visible light	Photopatch test
Polymorphous light eruption	NL / ↓	NL / ↓	NL	Negative
Chronic actinic dermatitis	↓	↓	NL / ↓	Negative / positive
Solar urticaria	Urticaria	Urticaria	Urticaria	Negative
Phototoxicity	↓	NL	NL	Negative
Photoallergy	↓	NL	NL	Positive

NL: Normal

1 Polymorphous light Eruption : PMLE

- most common type of photodermatosis
- abnormal Reaction to sunlight (UVB - UVA · rarely visible light)
- **It affect**: young adults, light complexion from may to October every year.
- **Clinically**:

1- eruption appear 4hrs - 4 days after sun exposure in the exposed areas :- Face - V area of chest, neck, arm

2. **4 Types present**:

- papular - papulovesicular - plaque - Diffuse Erythematous
  - ↓ Salty indurated Plaque → DLE

3- No Scarring atrophy

- **Juvenile Spring eruption**

- The helicles of the ears (Boys) - Their ears more exposed.
- with vesicles
- the tendency for eruption ↓↓ as the summer or sunny vacation proceeds "**Hardening**"

- **Histopathology** :- all types are non-specific except plaque Type :
patchy lymphocytic infiltrate → resembling DLE
But the patchy infiltrate is perivascular
Absent basal hydropic Degeneration

- **Diagnosis** :

+ve photoTesting : wave length < 320 nm
- ve lupus Band test → more important

- **Prophylaxis** : = Induction of Hardening
  1. initiated During spring
  2. 2-3 sessions weekly → of NB-UVB, PUVA (about 5 weeks
     + Oral prednisone (1mg/kg) During the initial 7-10 Days of the ttt → to minimize photoexacerbation
  3. ptn then asked to expose themselves to noonday sunlight for 15-20 minutes (without sunscreen) weekly for the remainder of the sunny season to → maintain the hardened state

- For NB-UVB: initial starting Dose = 50-70% the MED
  The Dose then ↑↑ By 10-15% per ttt
- For PUVA: 0.5-0.6% mg/kg of 8-methoxy-psoralen → given 1 hr before UVA exposure
  - The starting UVA Dose range 0.5-3 J/cm² ↓ Depending on Skin phototype.
  - The UVA Dose is ↑↑ By 0.5-1.5 J/cm² per ttt

- **Treatment:**
  - Topically: Steroids - Sunscreens
  - Systemic: Antihistamines, Systemic Steroids antimalarials

2 Actinic Prurigo

Hutchinson's Summer prurigo

- onset: appear During childhood. girls
- lesion: prurigo-like papules, small vesicles, pitted scars → present throughut the year
- Worst in summer involveing exposed and Covered areas
- frequent personal-family History of atopy

- Managment: photoprotection -PUVA NB-UVB -Calcineurin inhibitors

Actinic prurigo vs PMLE

Feature	Actinic prurigo	PLME
Starts	2-9 years of age	9-29 years of age
Relation to UV exposure	Often noted later	Clear
Pruritus	Severe, persistent	Transient
Covered areas of body affected	Frequent	Rare
Scar formation		
Ears affected		
Distal one-third of nose affected		
Plaques on the philtrum		Never
Cheilitis of the lower lip		
Conjunctivitis	Possible	Never
HLA association	DR4/DRB1*0407	None
Prophylaxis	Difficult	Easy

3 Hydroa Vacciniforme

- onset: Early childhood
- lesion: Deep seated umbilicated vesicles on exposed areas
- Healing: with Large variolifform scars
- E-Barr viral infection → detected in some pts
- Managment:
  - very careful photoprotection
  - B-Carotene - antimalarials - azathioprine Thalidomide - Cyclosprine Dietary fish oil

Chronic Actinic Dermatitis CAD

- photosenstive eczema (PE), persistant light reactivity (PLR), chronic photosenstive Dematitis (PD) → precursors of actinic reticuloid

- The Diagnosis of CAD depends on:

3
- 1-persistent eczematous eruption of sun-exposed skin with possible extension into non-exposed area
- 2- photosensitivity to UVB and often also longer wave length
- 3. Histologically → evidence of chronic eczema, with or without eczema-like changes

- **pathogenesis:**

- Transition from photoallergy to persistant Light reactivity
- alteration of some normal skin Components During photoallergic reactions Become → antigenic on its own.
- If CAD supervenes → UVB irradiation may Trigger the Delayed-type hypersensitivity Response at any site By: Formation of an antigenic photoproduct from the endogenous Carrier protien alone or in the absconce of exogenous initiating agents.
- +ve patch or photopatch test → common

- **Managment:**

→ By:- photoprotection
- avoidance of (photo) Contact sensitizer
- intermittent oral and Topical Corticosteroids
- Topical tacrolimus
- Low-Dose PUVA
- Cyclosporine
- azathioprine
- Mycophenolate Mofetil

→ Probability of spontaneous resolution is 10% over 5yrs
9 20% over 10 yrs

5 Actinic Reticuloid

- lesion: severe persistant photosensitivity with: Erythema, edema, Striking "Leonine" thickening of the light-exposed skin of the face, neck, hands
- prone: photosensitive atopic Dermatitis → more to develop actinic Reticuloid
- Occur: almost exclusive in elderly men
- There are: lichenified plaques first in the exposed area
  Later: The eption gradually spreads to cover most of the skin surface.
  ↓
  Erythroderma + generalized LN enlarge
- There is ↑↑ Thickening (deep Furrows) and Hyperpigmentation of the exposed areas
- Itching → severe
- The action spectrum → is UVA

- Histopathology:
  - Band like infiltrate of lymphoid and histiocytes in upper Dermis → extend into Lower dermis or invade into epidermis → aggregates → resembling "pautrier microabscesses"
  - Benign - reversible → T-cell in Skin lesions and circulation are the suppressor (OKT-8) Type ↓
    - where in CTCL → its of helper (OKT-4) Type
- Treatment:
  - systemic steroids
  - PUVA
  - Azathioprine
  - Sunscreens → ineffective

Leonine facies - associated dermatologic disease

- Scleromyxedema
- systemic amyloidosis
- lipoid proteinosis
- lepromatous leprosy
- leishmaniasis
- Cut lymphoma
- leukemic cutis
- Mastocytosis
- Sarcoidosis
- Actinic Reticuloid form of chronic actinic Dermatitis
- pachydermoperiostosis

6) Solar Urticaria:-

- physical urticaria → induced By :- Sun exposure
- lesions appear within 5-10 minutes and resolve over 1-2hrs.
- Chich By : immediate urticarial Respone to sunshine
- occuring : in sun-exposed skin.
- Systemic symptoms: ocur if there is sufficient mast cell release.
- The action spectrum generally → includes:- Both UVA, visible light rarely UVB.
- slight female predominance
- with onset at any age -
- Treatment: → plasmapheresis - IVIg
→ avoidance - sunscreens
→ High Dose - non sedating antihistamines
→ PUVA → Cause depletion of mast cell Contents

Classification of solar urticaria

	Mechanism	Action spectrum
Type I	Allergic	UVB
Type II	Unknown	UVA
Type III	Unknown	Visible light
Type IV	Allergic	Visible light
Type V	Unknown	UVA, UVB, visible light
Type VI	Protoporphyrin	Visible light

- pathogenesis:
- Chromaphore → present in the skin or in the circulation, OR Both may absorb radiation
- photoallergen formation
- IgE mediated Hypersensitivity to this photoallergen then develops with mast cell activation.

7) Solar (actinic) elastosis:

- ocur after prolonged exposure to Sun rays. Specially: fair-skinned persons.
- Small yellowish papules - plaques → Develop on Face and Back of Hands.
- The skin show: Deep furrows and wrinkles
- Subject to Development of actinic Keratoses and Carcinomas

- **Cutis rhomboidalis nuchae:**

- The skin on the Back of the neck → Become thickened and tanned with exaggerated normal skin markings

- **Histopathology:**

H & E

- upper dermis → Basophilic degeneration of the collagen
- Separated from the atrophic epidermis By narrow Band of Normal Collagen

By elastic tissue Stain

- aggregates of Thick, interwaven Bands of elastotic material which is newly formed by: fibroblasts → which are no Longer Capable of producing normal elastic fibers or collagen

8 Nodular elastosis with Cysts, Comedones:-

[Favre-Racouchot Syndrome]

- some pts è pronounced solar elastosis of the facial skin show: especially lateral to the eyes → multiple comedones - as well as → Yellowish nodules, Contain: central comedo.
- Variant of nodular elastosis:- è Cysts and Comedones is the "**actinic comedonal plaque**" → its found as: Solitary plaque on - sun damaged skin of either the arms or the face
- The plaque show: small nodules - Dilated follicles

- **Histopathology:**
  - pronounced solar elastosis
  - Dilated pilosebaceous openings
  - Large - round - Cysts → lined By: flattened epidermis and Represent: greatly extended Hair follicles.

Photosensitivity

- number of substances known as photosensitizers → induce :- abnormal reaction in skin exposed to sunlight
- These photosensitizers come in Contact è the skin Through → external or internal routes

I) Phototoxic reactions :-

- ch-ch By : exaggerated sunBurn-like reaction.
- Caused By : Systemic agents.
- The Cutaneous porphyrias : are an example of
  ↳ phototoxicity induced By endogenous agents.
- It is non-immunologic Reaction Can be elicited in the majority of 2 individuals 2-6 hrs after exposure of the photosensitizers →

- The Cytotoxic effect are Due to generation of-
  1- O_2 free Radicals - superoxide anions - hydroxyl radicals
  2- stable photoproducts (è chlorpromazine, tetracyclines)
  3- photoadducts (è psoralens)
  4- Inflammatory mediators (è porphyrins)
- Phototoxic Drug Reaction :-
  Severe sunburn without itching e.g: è sulfonamides, psoralen, Doxycycline
- phototoxic Contact Dermatitis :-
  - with Coal tar derivatives and psoralens as : OiL of bergamot → which are present in some perfumes → "Berloque or perfume Dermatitis" → manifested By streaking or drop-shaped lesions on sides of neck

II) Photoallergic reactions :-

- photallergy presents with :- eczematous lesions, related to topical photoallergens
- iTs a cell mediated - Delayed immunologic response Can elicited only in small number of individuals who

have been sensitized By previous exposure to photosensitizer Drugs and at the same time to light as in allergic contact dermatitis

- the role of light consists in: altering either the hapten itself or the avidity with which the hapten → Combines with the Carrier protien → to Form Complete photoantigen.

- photoallergic Drug eruption Causes a: photocontact Dermatitis in all light-exposed areas.

- il- Cause → Itching

- **photoallergic Drug reactions:**
  - e.g: Chlorothiazides, tolbutamide
    phenothiazines (chlorpromazines)
- **photoallergic Contact Dermatitis**
  sulfathiazides . antihistamines

- **Histopathology:-**
  - ① phototoxicity: Scattered necrotic Keratinocytes 'sunburn cells' • Dermal infiltrate of primarily < lymphocytes / neutrophils
  - ② photoallergy:
    - Epidermal spongiosis + Dermal Lympho-histiocytic infiltrate
    - Indistinguishable from other Causes of spongiotic Dermatitis

- **Treatment of photosensitivity:-**

1- avoiding the offending agents → if Not possible
2- strict photoprotection (UVB-UVA) → required
3- for phototoxic reactions → analgesia helpful
4- Topical corticosteroids, for severe flares →
   Short courses of systemic corticosteroids
   ↓
   Can be used for photoallergic Dermatitis
5- evening dosing of phototoxic Drug →
   Can be Done so that peak systemic levels occur During the night.

phyto.photo.dermatitis

- it's a: Form of phototoxicity Caused By: The Combination of a Topical or oral photosensitizing agents.
  [furocoumarins "psoralens .angelicins → most common agents]
- followed By: exposure to the appropriate wavelength of UV radiation.
- Limes - Celery - rue → the most common Causes
- it's Not an immunologic Reaction → So No prior sensitization is necessary and anybody can be affected.
- photoallergic reaction → to a plant is exceedingly rare.

Clinical Features :-

1. Cut-sensitivity to UV light peaks 30-120 minutes after contact with furocoumarins
2. Bizarre configurations of Erythema, edema and bullae → appear after 24 hrs, peak 72 hrs.
3. These painful, non-pruritic Reactions → more often seen in mid to late summer when psoralen concentrations are highest in the offending plants, more skin is exposed to Direct sunlight.
4. Hyperpigmentation appears 1-2 weeks later and lasts months to years.
5. Low-Dose UVA or psoralens → Cause:- Hyperpigmentation, without a preceding Vesicular or erythematous eruption.

Treatment :-

1- prevent Contact e offending plants
2- if Contact occurred → prompt washing with soap and water → may prevent a reaction

	• Solar urticaria • Erythropoietic protoporphyria
Papule	• Polymorphous light eruption • Actinic prurigo • Chronic actinic dermatitis
Vesicle	• Polymorphous light eruption • Juvenile spring eruption • Porphyria cutanea tarda • Variegate porphyria • Coproporphyria • Phototoxicity • Photoallergy • Hydroa vacciniforme
Erosion, crust	• Actinic prurigo • Hydroa vacciniforme • Porphyrias (PCT, VP, CEP, HC)
Eczema &/or lichenification	• Chronic actinic dermatitis
Erythema	• Phototoxicity
Scars	• Hydroa vaccinforme • PCT • VP • CEP

CEP: Congenital erythropoietic porphyria, HC: Hereditary coproporphyria, PCT: Porphyria cutanea tarda, VP: Variegate porphyria.

& action spectra of photosensitive skin diseases

	Spectrum of light		
	UVB	UVA	

Photoprotection : Sunscreens

II SunScreens:

- a preparation that attenuate UV wavelengths That would interact with molecules in the Skin (chromophores) → To generate → photochemical Reactions ⟹ photobiologic changes

Mechanism :-

- sunscreens forms a film or coat on the surface of the stratum corneum

absorb the photons reaching the Skin
- The energy absorbed By the organic agents → Converted to → non-Damging energy and dissipated primarily as heat

Scatter the photons Reaching The Skin
Scattering of Radiation is based on particle Size

Combination of Both Types
offering High levels of protection against Both UVB and Longer wavelengths

Sunscreens Applications :-

1- applied ½ hr before sun exposure
2- The proper quantity of sunscreen to Cover The Skin of an adult is 30 mL or 2 tablespoon
3- strategies to improve Compliance:-
- applying the product twice → to gain the proper Concentration → Reducing the occurrence of "Skip" areas
or use a product with twice the SPF desired

Active ingredients : in sunscreens

1- physical:
- e.g : Titanium dioxide, Talc, Zn oxide
- which act as reflectors and scattering agents
- Form an opaque barrier to all sunlight But They are Cosmetically Unacceptable
- Can discolor clothes
- may promote miliaria-folliculitis
- limited protection when a persons sunbaths for hrs

2-Chemicals:

- non opaque - colorless - Contain absorbing chemical

e.g :-

* para-aminobenzoic acid PABA and PABA esters :-
  - penetrate st. Corneum → where it can remain attached to proteins Through hydrogen bonding.
  - it can give protection even after Bathing, Swimming, perspiring
  - PABA → Highly effective UVB absorbers
* Benzophenones : Oxybenzone

They absorp UVB and shorter wavelength

* Cinnamates : They absorb mostly UVB and some UVA
  - They Don't Bind to St. Corneu so more easily Removed
* Dibenzoylmethanes : more effective UVA screen But poor UVB absorber

3-other agents :-

* Dihydroxyacetone : compound most commonly used in Sunless tanners
* Iron Oxide : offers protection across the UV spectrum and into the visible range, used as a colorant.
* othe active ingredients
  - added to sunscreens → with claims of ↑↑ efficacy of the finished products e.g: Antioxidants, vit E, C, green tea
* Boosters:

They are Not UV filters, But they serve to ↑↑ the protectiv nature of sunscreen Example: Tiny spheres : Scatter incoming radiation and agents that ↑↑ the spread of the sunscreen on the skin ⇒ greater production

Efficacy of Sunscreens :-

- Depends on: vehicle, pH stability - SPF - Type of skin evironment - sweating - Substantivity (The sunscreen Remain effective after prolonged sweating or swimming)

Determination of the sun protection factor (SPF)

- **Instrumentation:** Light source which mimics solar spectrum.
- **Procedure:** Determine minimal erythema dose (MED) in protected* & unprotected skin.
- **SPF =** $\frac{\text{MED protected}}{\text{MED unprotected}}$
- **To test substantivity** – after application & before MED testing:
  - Water resistant (40): 2 x 20 min water immersions (whirlpool bath)†.
  - Water resistant (80): 4 x 20 min water immersions (whirlpool bath)†.

- a product e` SPF of 10 → would allow 10 times as much time in the Sun e` The same Resultant level of Erythema as without The product in agiven individual
- an SPF 20 Similary would allow 20 times as much exposure with The same Result But → this doesn't mean that the SPF 20 product absorbs twice as much Radiation as the SPF 10 products

Sun protection is adviSed To :

- Fair skin (Type I-2)
- Sun senstivity disorders
- Personal or family History of skin cancer
- outdoor occupations

Labeling of sunscreens – ultraviolet B (UVB) and ultraviolet A (UVA) protection (2011)

Ultraviolet B (UVB) protection
• Sun protection factor (SPF): Up to 50+
Ultraviolet A (UVA) protection
• Either no label or broad spectrum.
• Broad spectrum can be used if critical wavelength is ≥ 370 nm.
Substantivity
• Water resistant (40 minutes) or water resistant (80 minutes).
• No use of the following designations: "Waterproof", "sunblock", "all day protection", or "sweat proof".
Additional labeling
• If a sunscreen has SPF ≥15 and is broad spectrum, then can state that the product can help to reduce the risk of skin cancer & the risk of early skin aging, when used regularly & as directed in combination with other sun protection measures.
• If the SPF of the sunscreen is <15 or it is not broad spectrum, then it must have the following skin cancer/skin aging alert: "Spending time in the sun increases your risk of skin cancer & early skin aging. This product has been shown only to help prevent sunburn, not skin cancer or early skin aging".

Adverse effects of sunscreens:

- minor skin irritation → common
- Allergic contact Dermatitis → Rare
- Effect on vitamin D
  - sunscreens that are effective at Blocking UVB photons → may Block some Cut. vit D synthesis
  - a Diet that include vit D rich Food and moderate amount of supplements Combined w modest amount of everyday sun exposure → is enough to maintain adequate serum vit D level → even if the individual photoprotects w Sunscreen.

II Systemic photoprotections

e.g:- Beta. Carotenes
antimalarials

Diseases Caused or aggrevated By Cold

① Reaction to extreme Cold

- Frost Bite: occur when the skin tempriature Drops Below about -2°C
- Clp:-

-Erythema - edema - numbness → Replaced By Maiked hyperemia , pain

-Occuring mainly on : Exposed parts (ears - nose - fingers - toes)

-Tissue Freezing, Vasoconstriction and inflammatory mediatorers Release (Pathophysiology) ↓

- Treatment → Rapid Rewarming in warm water Bath is the Cornestone

⑪ Abnormal Reaction to Cold

- Chilblain (pernio)
- Acrocyanosis :- persistant Dusky discoloration of Hands , feets with coldness in young girls
- Erythrocyanosis
- Livedo - Reticularis :- Cyanotic discoloration of skin & network pattern
- Raynaud's disease : also in LE, sclerodema Dermatomyositis - RA , Cryoglobulmemia
- Cold Urticaria
- Cryoglobulinemia
- Neonatal cold injury or sclerema
- Cold panniculitis

Pernio chilblains

-D.F: abnormal inflammatory Response to cold, damp, non-freezing Conditions

-Pathogenesis : 1-Vascular Origin

2- in children → associated ē Cryoglobulins or Cold agglutinins

-Epidemiology:

- Exposure to cold - wet conditions specially in area where Homes lack Contral heating
- women, children, the elderly are most Comonly affected
- Elderly ptn may Have prolonged course - younger ptn → improve spontenously.

- clinical Features:

1- single or mullple Erythematous to Blue-violet macules, papules, nodules
2- In sever Cases → Blistering - ulceration ocur
3- Bilaterally Symmetrically on the distal Toes - fingers, less often on the heels, nose, ears
4- Deep pernio: affect thights, Calves and Buttocks, Blue-erythrocyanotic plaques
5- Symptoms: Itching, Burning or pain
6- lesions often Resolve in 1-3 weeks except among elderly ptn è venous insufficiency

-pathology (non specific

- Dermal edema + superficial - deep lymphohistiocytic infiltrate è peri-eccrine accentuation
- Necrotic Keratinocytes - lymphocytic Vasculitis

-Laboratory evaluation:-

1- CBC → to exclude hemolytic anemia and myelomonocytic leukemia
2- Cryoglobulin, Cold agglutinin and Cryofibrinogen Levels to eliminate Cold-Senstive dysproteinmia
3- Serum protien electrophoresis and Immunofixation electrophoresis to exclude a monoclonal gammopathy

-treatment:

1- adequate Clothing - avoidance of cold - damp Conditions
2- Avoidance of Smoking

3-Nifedipine
4-Nicotinamide, phenoxybenzamine
Sympathectomy, erythemogenic
UVB phototherapy

Erythema ab igne

→ prolonged exposure to moderate Heat → emanating from fireplaces, radiators, heating pads
Causes → persistant Reticular Erythema with or without pigmentation
→ the shins - Buttocks → most common site
→ possible Development of → Cutaneous Sq. Cell Carcinoma or Merkel Cell Carcinoma

Radiodermatitis

- The effect of ionizing Radiation on the cells depends upon the amount and intensity of Radiation and the type of individual cell.
- These effects vary from arrest of mitosis, chromosomal Damage.
- Cell Death

Early acute

- after Large Doses of X-radiation or Radium
- Erythema develops within about week.
- heal & desquamation, pigmentation
- If dose High enough → Painful Blisters develop.
  → Healing & atrophy - telangectasia, irregular Hyperpig.
- Very Large Dose → ulceration occur

late (chronic)

- occur from few months to many years after the administration of fractional doses of X-rays or Radium
- Skin → atrophy · telangectasia, irregular hypo-Hyperpigmentation
- Ulceration - foci of Hyperkeratosis

Immersion Foot

- Injury → occur after continuous exposure of Feet to moist, occluded conditions
- Cold water, warm water - tropical variants exist
- The underlying pathological process → over-hydration of St. Corneum
- Neuropathy may persist indefinitely
- Feet exposed to immersion injury → are more sensitive to Re-injury
- prevention is the Best ttt.